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Environmental Assessment

Waste Treatment Facility

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HUMAN HEALTH AND SPECIAL WASTE

Prepared for

Ontario Waste Management Corporation

by

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Washington, D.C. and Princeton, N.J.

November 20, 1986

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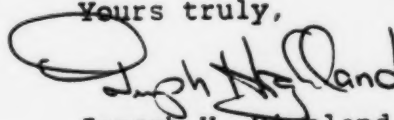
November 20, 1986

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Dear Sir:

We are pleased to submit our report entitled "Human Health and Special Waste", which provides information on the health effects associated with contaminants in special waste and their inadequate management.

Yours truly,



Joseph H. Highland, Ph.D.
Principal

LMT:slh

Enclosure

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EXECUTIVE SUMMARY

PURPOSE OF THE REPORT

The purpose of this report is to identify the human health effects that may be associated with exposure to special wastes in order to provide a basis for establishing that there exists a need for proper treatment and disposal of these wastes.

SCOPE AND METHOD

In this report, the potential impact of inadequate disposal of special wastes on human health is analyzed through a general discussion of the following topics:

- Adverse Health Effects Associated with Chemical Exposure -- Major human health effects associated with exposure to constituents of special wastes are reviewed. End points considered include organ damage, cancer, genetic defects, reproductive abnormalities, immunological imbalances, nervous system disorders, and disorders of the skin and blood.
- Assessment of Health Effects -- Methods for assessing the relationship between chemical exposure and human health effects associated with disposal sites are reviewed. The strengths and limitations of the various methods reviewed are discussed.
- Evidence of Human Health Effects Associated with Improper Disposal of Special Wastes -- Selected case studies are presented, which indicate that the inadequate disposal of special wastes has resulted in the release of contaminants to the environment, human

exposure to the contaminants, and consequently adverse human health effects. Specific examples include disposal sites in Hamilton, Ontario; Love Canal, New York; and Hardeman County, Tennessee.

STATUS OF DATA

Data concerning human health effects associated with exposure to specific chemicals have been compiled by ENVIRON through a review of the relevant literature. Information on case studies involving the improper disposal of special wastes has been derived from published accounts. In some instances, the conclusions reached in these accounts have not been critically evaluated by ENVIRON or cross-checked against other possible sources of information.

MAIN FINDINGS

The main findings of this report include:

- Exposure to chemicals present in special wastes may result in a variety of adverse human health effects.
- Despite limitations, various methods can be used to assess human health impacts associated with exposure to environmental contamination from chemicals released from disposal sites.
- Case studies have indicated that the improper disposal of special wastes can lead to a variety of adverse health effects in exposed populations.

CONCLUSIONS

Laboratory studies have shown that human exposure to constituents of special waste can result in a variety of adverse health effects, ranging from minor irritation to serious injury or death. Although cause-and-effect relationships are difficult to establish, there is a large body of evidence to strongly suggest that exposure to chemicals released from disposal sites may cause adverse health effects in surrounding populations. Thus, there exists a need to establish waste management facilities that minimize the release of special waste to the environment and therefore reduce the potential for human exposure to such waste.

I. INTRODUCTION

Waste is any material that is no longer wanted and has no current or perceived future value. Ontario Waste Management Corporation (OWMC) has defined liquid industrial waste and hazardous waste as "waste that requires special treatment". These special wastes have physical, chemical, or biological characteristics that can, under certain circumstances, place human health at risk or adversely affect the natural environment. Special wastes can be solids, liquids, or gases and are byproducts of a wide variety of manufacturing and service industries. Table 1 presents some common consumer products and the types of wastes their manufacture produces (Goldman et al., 1986). A study to determine the quantities and types of special wastes generated by Ontario's manufacturing industries has been conducted by OWMC (DPA, 1986).

The potential adverse health effects in humans exposed to special waste constituents in the environment are dependent upon many factors. The ultimate health effect depends on the toxicity of the chemical, the dose reaching the target organs, the route

TABLE 1
COMMON PRODUCTS AND POTENTIAL HAZARDOUS WASTES
GENERATED DURING MANUFACTURING

<u>Products</u>	<u>Potential Hazardous Wastes</u>
Plastics	Organic chlorine compounds, organic sludges
Pesticides	Organic chlorine compounds, organic phosphate
Medicines	Organic solvents and residues, heavy metals such as mercury and zinc
Electrical Equipment	Cyanides, heavy metal sludges, caustics, solvents, acids, polychlorinated biphenyls (PCBs)
Paints	Heavy metals, pigments, solvents, organic residues
Petroleum Products	Oil, phenols and other organic compounds, heavy metals, ammonia salts, acids, alkalis
Metals	Heavy metals, fluorides, cyanides, acid and alkaline cleaners, solvents, pigments, abrasives, plating salts, oils, phenols
Leather	Heavy metals, organic solvents
Textiles	Heavy metals, dyes, organic chlorine compounds, solvents

Source: Goldman et al., 1986

of entry, interactions between the chemical and other concurrent chemical exposures, and the normal protective and restorative mechanisms of the body. Chemical exposures occurring over a short duration are termed acute exposures, and generally are associated with immediately observable effects. These effects may be reversible and cease when exposure ceases, or they may persist beyond the period of exposure. Other types of chemical exposures occurring at low doses over a long period of time (chronic exposure) do not produce immediately observable effects; it is only after a relatively long period of exposure that adverse health effects (chronic effects) result. Chronic exposures are most frequently associated with chronic effects such as cancer.

It is desirable to have an understanding of the potential adverse effects that may be associated with chemical exposure and of the methods available to assess the incidence of disease in populations exposed to chemicals released from disposal sites. This information is presented in the following chapters:

- Chapter 1, the introduction,
- Chapter 2, a discussion on adverse health effects associated with chemical exposure,
- Chapter 3, a discussion on methods for assessing health effects in populations exposed to chemicals released from disposal sites,

- Chapter 4, a presentation of case studies documenting the development of adverse health effects after exposure to improperly disposed of special wastes, and
- Chapter 5, conclusions.

II. ADVERSE HEALTH EFFECTS ASSOCIATED WITH CHEMICAL EXPOSURE

A. INTRODUCTION

To establish a basis for understanding the nature of the potential human health effects associated with exposure to a specific chemical, available toxicological studies are evaluated. The most relevant data are from studies in humans, which are generally available from accidental or occupational exposure. For example, evidence of the carcinogenicity of vinyl chloride and asbestos comes from studies of workers exposed to these substances in the work place. Similarly, the evidence linking adverse health effects with exposure to hazardous benzidine dyes also comes from occupational studies. However, the availability of human data is limited and thus studies involving laboratory animals are most often used to evaluate toxicological effects.

Animal toxicity studies are designed to identify the nature of health damage produced in an individual by exposure to a specific chemical substance and the range of doses over which damage is produced (dose is often defined as the weight of the chemical agent received daily by an exposed animal divided by the

weight of the animal). Toxicity investigations usually begin with a study of the acute (single-dose) toxicity of a chemical in experimental animals, in order to calculate doses that will not be lethal to animals used in toxicity studies of longer durations. These studies also provide an estimate of the comparative toxicity of the compound and may indicate the target organ system that will manifest chronic toxic effects. After the acute toxicity of a chemical has been investigated, subchronic (repeated or continuous exposure for several weeks or months) or chronic toxicity studies (exposure for close to the full lifetime of the experimental animal) may be performed to learn how the period of exposure affects toxic response.

The use of animal toxicity data to predict potential toxic effects in humans is based on several assumptions about the biological similarity of various mammalian species, as well as assumptions concerning the applicability of toxicity test conditions (exposure route, doses, etc.) to human exposure conditions. The use of laboratory studies in assessing the risk to humans of exposure to toxic chemicals includes the following limitations (Grisham, 1986):

- The route and nature of exposure used in laboratory studies may not be similar to human experience.

- Species differences in absorption or metabolism may result in species differences in toxicity.
- The methods commonly employed entail extrapolation of effects in animal systems from the large doses used experimentally to the much smaller doses likely to be encountered in environmental exposures.
- Suitable animal models may not be available for assessing specific human health effects.

Even given these limitations, it is generally assumed that human toxicity can be inferred from observations in experimental animals unless there are biological reasons to consider certain types of animal data irrelevant to humans. In fact, experimental animal data often form the basis for regulatory policy. While laboratory investigations of species other than humans are generally considered adequate for inferring causal relationships between exposures to chemicals from disposal sites and adverse human health effects, this evidence alone may not be sufficient for establishing conclusively that an exposure to a chemical will result in a specific type of effect. For some human health effects, no suitable animal model exists and thus evidence can only be derived from direct observations of exposed people (Grisham, 1986).

The potential adverse health effects that may be manifested in populations exposed to hazardous substances can involve impacts on any body function or organ system. The nature of the

health effects depends on the specific chemicals contacted, the extent of exposure, the characteristics of the exposed individuals (i.e., sex, genetic background and age), the metabolism of the chemicals involved, and the presence or absence of other variables such as other diseases or concomitant exposures. Health effects of primary concern to populations exposed to hazardous chemicals include organ damage, cancer, genetic defects, reproductive abnormalities, immunological imbalances, nervous system disorders, and adverse impacts to the blood and skin. These health effects are discussed separately below.

B. ORGAN DAMAGE

1. Liver

A major function of the liver is to aid in metabolism, i.e., the biochemical conversion of one substance into another for purposes of nutrition, storage, detoxification, or excretion. Multiple mechanisms exist within the liver for each of these processes, and interference can lead to a toxic effect. Chemicals that cause damage to the liver are termed hepatotoxic. Toxic endpoints of the liver include lipid (i.e., fat) accumulation, jaundice, cell death (necrosis), and cirrhosis (hardening). In addition, chemicals that increase the level of liver metabolic

enzymes, i.e., enzyme inducers, can dramatically affect the toxicity of other compounds by altering their metabolism. Some classic examples of hepatotoxic agents are carbon tetrachloride, chloroform, and trichloroethylene, all of which have been used as industrial solvents and are present in special waste generated in Ontario. In addition, chemicals such as PCBs, beryllium, arsenic, and vinyl chloride also exhibit hepatotoxicity and are present in special waste.

2. Kidney

As an organ whose major function is the elimination of toxic substances and other waste products, the kidney can be considered a complex, elaborate blood filter. The kidney concentrates wastes for elimination and retains nutrients and water that are useful to the body. In addition, the kidney can metabolize and detoxify some of the same chemicals as the liver, although the rate of metabolism is usually slower. Chemicals known to injure the kidney are called renal toxicants. Examples of renal toxicants that are present in special waste include cadmium, mercury, chlorinated dibenzo-p-dioxins, DDT, PCBs, hexachlorobenzene, carbon tetrachloride, and chloroform. Some renal toxicants may cause cell death. However, because the kidneys must process large volumes of blood, they are first susceptible to toxic damage that interferes with the flow of

blood and urine. This change can be mechanical, e.g., due to the presence of kidney stones or due to the presence of chemicals that dilate or constrict passages that would affect the rate of blood filtration and urine production.

3. Lungs

The major function of the lungs is to exchange oxygen and carbon dioxide between blood and air. This exchange can facilitate entry and exit of other substances from the body. Damage to the lung includes irritation and constriction of air passages, cell death, excessive fluid retention (edema), loss of elasticity of lung tissue (fibrosis), and decrease of effective surface area for gas exchange (resulting from breakdown of small air sacs). Exposure to chemicals that irritate the lung, such as ammonia and chlorine, can lead to a range of effects, from respiratory discomfort to death. Although the effects of acute exposure to irritants are usually reversible, chronic exposure may lead to permanent cell damage. The normal, necessary exchange of gases across the lung can be impaired by substances that constrict the respiratory passages, affect secretions or other normal functions, or physically remain in the lung. Substances, such as aluminum dust, coal dust, iron oxides, and ozone that cause necrosis, edema, or fibrosis will impair lung function. Exposure to certain substances, such as asbestos and arsenic, can lead to impaired lung function and cancer.

C. CANCER

Cancer is a term used to characterize the abnormal growth of cells that invade normal tissues or spread to distant organs. Chemical carcinogens are substances that have been found to induce abnormal, uncontrolled growth of cells in laboratory animals or in humans. Carcinogenesis--the induction and formation of a tumor--appears to be a multistage process requiring an initiating "event" (believed to be a genetic alteration in a cell) followed by proliferation of the affected cells into a tumor. A substance that is capable of initiating cancer by altering the genetic material of a cell is referred to as an "initiator." Due to the ability of most cells to repair damage to genetic material, not all "initiated" cells will progress to form tumors. In general, a substance that increases the incidence of tumors only when exposure follows initiation is called a "promoter." In contrast to the effects of initiators, the effects of promoters appear to require continual exposures over a prolonged period of tumor development and are reversible upon cessation of exposure. Both promoters and initiators can be distinguished experimentally from complete carcinogens--agents with both initiating and promoting capabilities.

The International Agency for Research on Cancer (IARC) has concluded that more than 140 chemicals are definitely

carcinogenic in laboratory animals and that "it is reasonable to consider them carcinogenic in humans" (as cited in Grisham, 1986). The United States Environmental Protection Agency's Carcinogen Assessment Group has identified 55 chemicals as being suspect human carcinogens (USEPA, 1985). These chemicals include some common constituents of special waste such as metals (e.g., arsenic, cadmium, chromium), pesticides (e.g., aldrin, chlordane, DDT, dieldrin), solvents (e.g., carbon tetrachloride, benzene), PCBs and tetrachlorodibenzo-p-dioxins (TCDD).

D. GENETIC DEFECTS

The integrity of the genetic material in all cells is critical to cell function and may be affected by some toxic agents. Genetic damage can occur by several mechanisms. The results are generally classified in three groups: mutations, clastogenic events, and aneuploidy. Mutagens are defined as substances that change the chemical structure of DNA. Since DNA is "read" to provide information necessary for cell function and proliferation, mutations may cause a misreading, leading to improper functioning of the cell. Clastogens cause a break in one or more strands of DNA and a physical rearrangement of the parts. Aneuploidy is an addition or deletion of the number of chromosomes; a commonly known type of aneuploidy is Down's syndrome in which there is an extra copy of a particular

chromosome in each cell of the body. Aneuploidy is often caused by chemicals that affect cell division.

Chemicals causing genetic toxicity can have many effects. Genetic damage can lead to altered cell proteins and, therefore, to improper functioning of cells. This, in turn, may lead to cell death or cancer. If the damage is in the reproductive system, chemicals inducing genetic toxicity can cause reproductive failure; damage to germ cells may result in abnormal offspring. In some cases, genetic toxicity may be caused by exposure to a chemical, but no evidence of an adverse genetic effect may be detected. This can occur because much of the DNA in cells is inactive. For example, skin cells do not produce hemoglobin and no effect will be observed if instructions for producing hemoglobin are damaged in a skin cell. Such events are called silent mutations. Examples of chemicals suspected of causing genetic defects in humans are benzo(a)pyrene, dibenzanthracene, hexavalent chromium, and some forms of nickel, all of which are constituents of special waste.

E. REPRODUCTIVE ABNORMALITIES

Reproductive toxicity can occur in the male parent, the female parent, or their developing offspring at any of the numerous stages that lead to the production of a viable human being.

Toxic effects on the reproductive system can be classified into three general types: impaired ability to conceive (decreased fertility), failure of the fetus to survive (conceptus or fetal death), and production of abnormal offspring (teratogenic effects). Decreased fertility usually results from impaired production of the sperm or egg or altered biochemical and physiological conditions required for fertilization. Death of the fetus, whether at the early embryonic stage or later fetal stage, can be caused by a variety of factors including exposure to chemicals. Such chemicals are labeled embryotoxic and fetotoxic, respectively. Teratogenic effects (birth defects) include abnormal skeletal, muscle, or organ structure, mental retardation, metabolic malfunctions, and subtle malfunctions that may not be noticed during a normal life. Examples of chemicals that cause reproductive effects in animals or humans include PCBs, TCDD, DDT, trichloroethylene, 1,1,1-trichloroethane, vinyl chloride, benzene, naphthalene, phenol, toluene, and xylene, which are common constituents of special waste.

As previously mentioned, exposure of a pregnant mammal to a teratogenic substance can cause developmental defects and result in abnormal offspring. For the developing fetus to be exposed, the chemical must pass through two barriers: the mother and the placenta. If a given dose of a compound is sufficiently toxic to

kill or injure the mother, it will obviously adversely affect the developing embryo or fetus. Although this statement may seem trivial, the converse is an important principle in teratogenesis. The more insidious teratogens are those that affect the developing organism at concentrations that are significantly lower than those that affect the mother. Although the placenta was once thought to be a rather strong barrier, many chemicals have been found to cross it. Moreover, the placenta is not inert but is capable of metabolizing some chemicals into either more or less toxic substances or concentrating some compounds in the amniotic fluid. This metabolism may also affect the flow of substances across the placenta.

The teratogenicity of a chemical is determined by its intrinsic ability to affect the developing organism and by the magnitude, duration and timing of exposure during fetal development. The latter consideration--time of exposure to the potential teratogen--may determine not only which developing system is affected but also whether the compound will have any effect at all. For most developing systems there is a critical period, usually occurring between three and twelve weeks of gestation, during which the system is particularly sensitive to chemically induced abnormal development.

F. IMMUNOLOGICAL IMBALANCES

The chief functions of the immune system are 1) defense against infection and disease; 2) homeostasis, tendency to maintain equilibrium; and 3) surveillance, ridding the host of altered or mutated cells that could lead to cancer. Exposure to some environmental toxic agents may stimulate or suppress the immune system. Toxic chemicals may stimulate immune responses by several mechanisms such as triggering an allergic reaction or stimulating production of factors that can enhance specific immune responses. Exposure to toxic chemicals that suppress the immune response may result in an increased susceptibility to infection and to some forms of cancer. Chemicals shown to cause immune dysfunction in humans or animals are common constituents of special waste such as benzene, phenol, toluene, formaldehyde, chloroform, PCBs, polybrominated biphenyls (PBBs), polycyclic aromatic hydrocarbons (PAH), pentachlorophenol (PCP), TCDD, and metals such as arsenic, cadmium, chromium, lead, mercury, and zinc.

G. NERVOUS SYSTEM DISORDERS

Communication and the control of reflexes, movement, integration of sensory information, autonomic functions (e.g., breathing), and cognitive functioning are the major functions of the central nervous system (CNS). All of these functions can be impaired by

a group of toxic chemicals called neurotoxins. Damage to the nervous system can occur in the brain, spinal cord or peripheral nerves, and may involve nerve cell bodies, nerve fibers that extend through the body, the myelin sheaths that cover these fibers, and the nerve-nerve or nerve-muscle junctions. Damage to nerve cell function is often called neuropathy. Different types of neuropathies occur depending on the site and function affected. In addition, behavioral changes are often caused by damage to the central nervous system. While it is generally accepted that exposure to some chemicals can cause headaches, fatigue, or irritability, it is difficult in any specific situation to determine whether such common symptoms are caused by chemical exposure, lack of sleep, depression, or other factors. Although these symptoms may be mild and difficult to assess, they may provide an early warning of exposure to a toxic chemical. Some examples of neurotoxins are toluene, trichloroethylene, cyanide, acrylamide, DDT, arsenic, lead and some forms of mercury, all of which are common constituents of special waste.

H. OTHER ADVERSE EFFECTS

1. Skin

The skin provides a barrier between the organism and its external environment. It prevents loss of body fluids, aids in the

maintenance of a constant temperature, and prevents entry of many substances. However, the skin can serve as a route of entry for some toxicants. Chemicals that are absorbed through the skin may affect other systems. Similarly, chemicals that enter by other routes may affect the skin, e.g., skin changes caused by oral ingestion of arsenic. Irritation, rashes, and itching are common toxic reactions to skin exposures. Chemical photosensitizers may cause an allergic reaction that becomes more severe with continued exposure to light, or may increase the skin's susceptibility to UV damage. Folliculitis (damage to the hair follicles) and acne are other common skin disorders. Chloracne is a particular form of acne that can be caused by exposure to several chlorinated hydrocarbons, including PCBs and chlorinated dibenzo-p-dioxins. Some substances can change skin pigmentation. Skin keratoses (hardening or scaling) or ulcers are additional toxic responses. Finally, skin cancers may be caused by direct contact with some substances (e.g., soot) or exposure to other substances (e.g., arsenic) via other routes.

2. Blood

Transport of oxygen, carbon dioxide, and other materials is a major function of blood. The hematopoietic (blood producing) system, which includes organs and tissues that produce, transport, and filter blood, interacts with the cells of all

other systems. Toxicity can affect developing blood cells, existing cells, or the hematopoietic organs.

In the human and other mammals, blood cells are formed in bone marrow, the three major types of which (red blood cells, white blood cells, and platelets) are formed from a common precursor cell. Red blood cells carry oxygen and carbon dioxide between the cells and the lungs, white blood cells function as part of the immune system, and platelets are necessary for blood clotting. Chemicals toxic to bone marrow, such as benzene, can affect formation of these cells. Depending on the stage of development and type of cell affected, any or all of these three major types of blood cells may be affected resulting in an abnormal increase or decrease in production. A hematopoietic organ that may be affected by certain toxic chemicals is the spleen, which is responsible for removing old or damaged red blood cells from circulation. In addition to benzene, naphthalene, phenylhydrazine, and arsenic, which are commonly found in special waste, are known to be hematopoietic toxins.

III. ASSESSMENT OF HEALTH EFFECTS IN POPULATIONS EXPOSED TO CHEMICALS RELEASED FROM DISPOSAL SITES

A. INTRODUCTION

Although there is a substantial body of laboratory evidence to indicate that chemical exposures may cause adverse health effects in individuals, such evidence alone is not sufficient to conclude that adverse health effects experienced in populations exposed to toxic chemicals arising from chemical disposal sites are causally linked. The degree, nature and duration of exposure must be considered as well as synergistic and antagonistic interactions among chemicals to which exposure has occurred. Such information is often difficult to obtain. In order to evaluate the potential causal relationship between chemical exposure and the onset of disease, the health status of the exposed population is often compared with that of an unexposed control group to see if significant differences exist because of the chemical exposures that have occurred. Comparative investigations of this type are called epidemiological studies.

Epidemiological studies can be used to provide a direct basis for the assessment of risk whenever human health effects are suspected to result from exposure to environmental

contamination. Epidemiological studies involve a comprehensive review of the entire body of research data. If the body of data is extensive and of good quality, then epidemiological studies can provide a clear and reliable basis for evaluating the cause-and-effect relationship between exposure to toxic chemicals and the increased incidence of adverse health effects. More often than not, however, the data are sparse, and thus the conclusions that can be drawn are limited.

The assessment of health effects associated with humans exposed to hazardous wastes is a very complex problem. This chapter discusses several methods for assessing the relationship between health effects and chemical exposure and the limitations associated with each of these methods.

B. HEALTH EFFECTS AND CHEMICAL EXPOSURE

Epidemiology is the discovery and interpretation of disease patterns in human populations, the development of quantitative assessments of these patterns, and the characterization of the relationship between personal and environmental characteristics and the observed patterns. Epidemiologists rely upon various study methods and sources of data to examine the health status of populations. For example, vital statistics, birth and death

certificates, and medical records may be used to examine the incidence of congenital abnormalities and various disease states, and mortality rates.

Information on adverse human health effects potentially attributable to environmental chemicals is obtained from four sources: 1) summaries of self-reported symptoms in exposed persons; 2) case reports prepared by medical personnel; 3) descriptive epidemiology studies; and 4) analytic epidemiology studies. The first two sources are generally considered anecdotal data, which when considered alone, are of limited use in determining causes of disease.

Descriptive epidemiological studies involve the identification of the nature, magnitude and distribution of disease in populations, often by employing a correlational approach in which differences in disease rates in human populations are associated with differences in chemical exposures. The unit of observation is the population, not the individual. They are often useful in drawing attention to previously unsuspected problems and in generating hypotheses that can be further tested. In the absence of additional supporting data, they are generally of limited use in identifying cause-and-effect relationships.

In contrast, analytic epidemiological studies investigate individuals, and are the primary means for determining causal associations between specific environmental exposures and human disease. These studies test specific hypotheses using "observational" study designs. Observational study designs have generally been used because "experimental" designs (study designs used in animal studies in which randomization is used to ensure against biased allocation of study subjects to experimental and control groups) are impractical or unethical in humans when evaluating agents or exposures with potential adverse effects. One exception to this would be randomized clinical trials for drug testing. Analytical epidemiological studies include cohort studies, case-control studies, cross-sectional studies, and outbreak investigations.

Cohort Study

Cohort studies involve the evaluation of the disease experience of individuals exposed by choice or accident to a particular environmental agent as compared with similar nonexposed individuals. In a cohort study, the population exposed to a suspected agent (cohorts) and a nonexposed population (controls) are followed for a period of time to determine who develops or dies of the disease in question. Incidence rates (number of new cases of disease per unit of population per unit of time) or

mortality rates (number of deaths per unit of population per unit of time) are then collected for the exposed and nonexposed populations and compared. Frequently, incidence or mortality rates are not available on an appropriate unexposed group of persons for comparison with the exposed population. In such cases, readily available general population incidence or mortality rates are used. Cohort studies give a direct measurement of relative risk (the risk of developing a disease or an adverse outcome in the exposed population relative to the risk in the nonexposed population). There are several problems associated with cohort studies, including: 1) long follow-up times that may increase the cost of the investigation; 2) loss of the ability to conduct follow-up studies (i.e., people move away, die, or lose contact), thus decreasing the ability to detect the disease; and 3) the large sample size needed to detect an increase in the incidence of a rare disease.

Case-Control Study

In case-control studies, individuals with a particular disease (cases) are compared with similar but nondiseased individuals (controls) on their history of exposure to a suspected agent. In such studies, the proportion of cases with a history of exposure to the agent is compared with the proportion in controls. The relative risk cannot be measured directly in case-control studies

but must be approximated. These studies are particularly useful for studying rare diseases, or for investigating potential associations between a disease and a number of agents. Case-control studies often have certain limitations, however, including the unavailability of information about past exposures, bias of recall of past exposures and difficulties in obtaining appropriate control groups.

Cross-Sectional Study

In a cross-sectional or survey study, subjects are selected irrespective of their disease or exposure status. In such studies, disease and exposure are evaluated at the same time. Consequently, the time sequence of exposure onset and disease onset cannot be determined from these studies. Thus, although the data from a cross-sectional study can be compared in a manner similar to that described above for case-control and cohort studies, they cannot be used to establish the time sequence of exposure and disease necessary for drawing conclusions about the cause of a disease. Deviation from expected measurements and observations, however, may suggest the need for further investigation or intervention in high-risk populations or subgroups.

Outbreak Investigation

An outbreak investigation is one in which a group of people has been reported to have developed similar symptoms, generally acute, and such reports are clustered temporally and spatially. This type of study can be particularly effective in eliciting the nature and source of the exposure when relatively high levels of exposure to a chemical are required to cause acute health effects.

The epidemiological studies described above often use health questionnaires as tools for collecting the necessary information for performing the investigation. However, there are limitations associated with the use of health questionnaires. For instance, reporting accuracy may be influenced by an individual's ability to recall specifics about his health problems. Secondly, surveys of disease or symptom incidence test for a possible increase in rare events and generally rely on nonparametric statistical methods for analysis. Both of these characteristics reduce the statistical power of the study considerably. A third problem is that the latency period for some chronic diseases makes it difficult to correlate chemical exposure to disease. Surveys of disease depend on health damage that has already occurred (Paigen and Highland, 1982).

Alternative testing methods to the use of health questionnaires would ideally 1) depend on objective physical or biochemical measurements so that subjectivity or reporting accuracy becomes irrelevant; 2) yield quantitative values in order to increase the statistical power; 3) detect potential health risk rather than clinical disease; 4) use testing that is non-invasive and acceptable to the community, and 5) be sensitive, inexpensive, and use readily available expertise and facilities (Paigen and Highland, 1982). A number of tests meet these criteria with various degrees of success, including adverse pregnancy outcomes, growth of children, and tests of neurotoxicity (Paigen and Highland, 1982).

C. EVALUATION OF HUMAN HEALTH RISK

Certain criteria must be applied to epidemiological studies to evaluate cause-and-effect relationships between chemical exposures and health status. These include 1) the strength of the association observed -- the incidence of a disease is much greater in those exposed than in those not exposed, and the incidence increases with increased exposure,¹ 2) the

¹ In addition to a positive association, the relationship between chemical exposure and incidence of health effects is also judged in terms of statistical significance, that is, the determination that the association is unlikely to have occurred only by chance. The level of significance (the probability of detecting an association when one does not exist) is dependent on the investigator's needs and objectives and is influenced by the sample size of the test population.

consistency of the results in various studies -- observations can be replicated in diverse circumstances using different study methods and populations, 3) the presence of a plausible biological mechanism -- the disease in question might be expected to result from the exposure on the basis of existing scientific knowledge, 4) temporal relationship between exposure and outcome -- exposure precedes outcome, and 5) the specificity of the association -- the extent to which one can predict whether an exposure will result in a specific health effect. Confidence that an exposure is causally related to a disease improves as the strengths of these criteria are increased. The association between cigarette smoke and lung cancer is a good example of a cause and effect relationship meeting most or all of the above criteria.

Epidemiological studies of populations near chemical disposal sites are seldom able to determine with certainty that an exposure and a health effect are related. Information on magnitude, duration, and timing of exposure for each individual involved is rarely available. Measuring human exposure is further complicated by the variability of individual exposures. For example, some people spend a major portion of the day away from the disposal site; people move in and out of the exposure area; and employed people can be exposed to chemicals in the work

place as well as in the home. In the absence of specific data, exposure is often characterized using less specific information such as proximity of residence to the site and duration of residence.

Another obstacle in the attempt to link disease and exposure to an environmental contaminant is the prolonged induction or latent period between the start of exposure and the manifestation of the disease. The latency period associated with a disease can be influenced by a number of factors, including characteristics of the exposed individual and of exposure conditions. Relatively short latent periods will enhance the ability to establish a cause-and-effect relationship. Health effects that generally exhibit short latent periods include decline in fertility, congenital abnormalities and decline in pulmonary function. Health effects that exhibit relatively long periods include malignancies and vascular diseases (Grisham, 1986). Thus, epidemiological studies should be designed to have a sufficiently long follow-up period to account for the latency period of the health effect of interest in order to avoid incorrectly evaluating the observed health effects in the exposed population. Other limitations that may weaken or invalidate the results of epidemiological studies include small sample sizes, selection of common or non-specific disease endpoints, and subjects' knowledge of the study's goals.

Another concern when considering chemical disposal sites is the "lag time" between the point at which chemicals begin escaping into the environment and the time human contact with the chemicals may occur. That is, chemicals must be released into the environment and be transported through the soil, air, water or food chain before human exposure can occur. In some cases, the length of the lag period may range from months to decades depending on the nature of the waste chemicals and the type of containment used, the physical characteristics of the site, the attenuation capacity of the surrounding environment, and the routes of exposure for humans (Grisham, 1986).

Often, adverse effects to the ecosystem can provide the first indication that chemicals are being released to the environment such that human exposure can occur. These adverse impacts can include contamination of ground water or surface water possibly resulting in fish kills or disruptions in the ecological health of the aquatic systems, pollution-induced chlorosis and necrosis of plant and tree leaves, and effects of pollutants on wildlife resulting from direct toxicity, habitat modification, or food chain dynamics, such as the thinning of egg shells caused by the bioaccumulation of PCBs. Observations of plants and animals can provide information concerning the nature of the contaminants and their potential for widespread pollution and toxic effects in the environment and in humans.

IV. EVIDENCE OF HEALTH EFFECTS ASSOCIATED WITH IMPROPER DISPOSAL OF SPECIAL WASTES

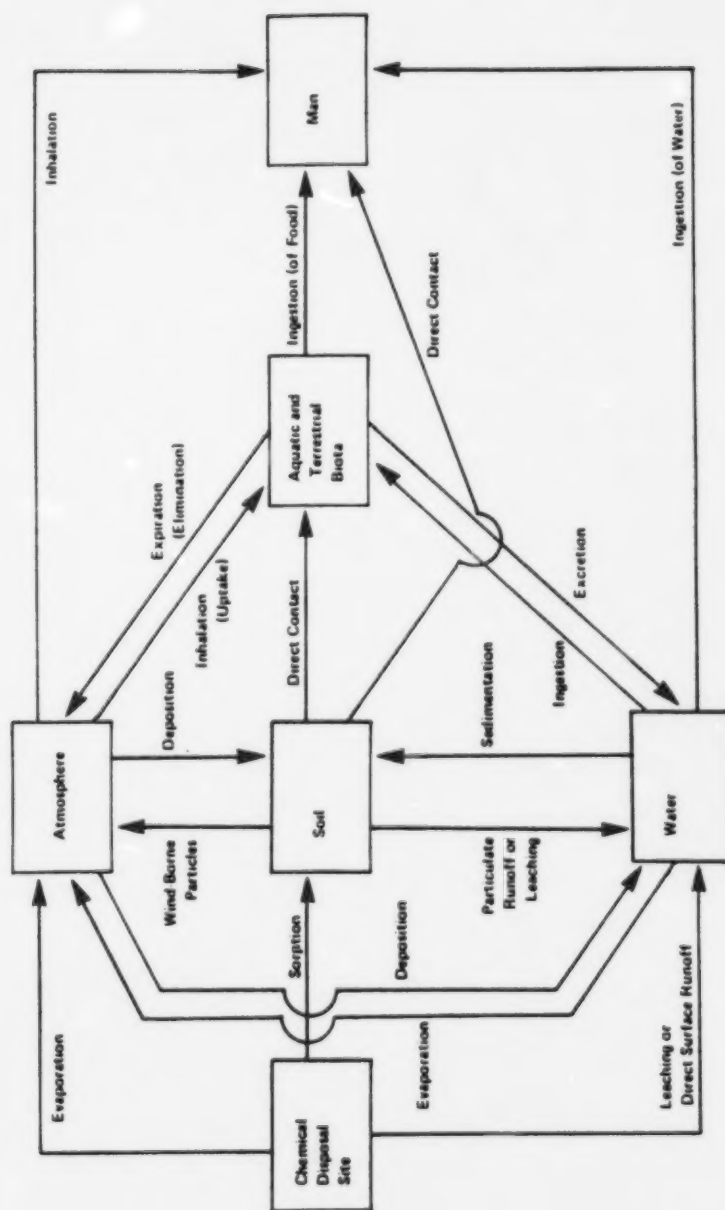
A. INTRODUCTION

Disposal of hazardous waste materials has historically been accomplished in the least expensive and most expedient manner possible, often by employing open dumps, landfills, bulk storage containers, or surface impoundments for untreated waste. Each chemical disposal site is unique with varying compositions of hazardous chemicals and differing geologic settings. The physical and chemical characteristics of the individual chemicals as well as the physical, chemical, and biological characteristics of the site environment influence the extent to which the chemicals are likely to move within a disposal site, be released, and migrate through the environment so that humans are potentially exposed. For example, chemical characteristics affect the decomposition of the waste materials, as well as interactions with other substances to produce new, and potentially more toxic, chemicals. Physical characteristics such as solubility and volatility influence the rate at which a chemical leaches into ground water or escapes into the air. The characteristics of the site environment, such as geologic setting, are important factors affecting migration of chemicals

from disposal sites. The nature of geologic material beneath the disposal site affects the movement of waste materials into the ground water. For example, clay soils retain many chemicals thus reducing the rate of release. On the other hand, sand and gravel favor rapid movement of liquid materials and thus do not provide good containment.

Management of the waste is also an important factor affecting release of the hazardous contaminants to the environment. For example, leaving hazardous wastes uncovered increases the chances for air emissions and direct contact of the waste, as well as increasing the potential for contamination of ground water due to leaching, and contamination of surface water due to flooding of the disposal area. A well maintained, low permeability cap placed over the site reduces the release of chemicals to the environment.

Chemicals released to the environment from the disposal site can reach humans through a variety of media such as ground water, surface water, air, soil and food. Figure 1 illustrates various pathways of chemical transport that may result in human exposure. Ground-water sources contaminated with toxic materials present a long-term threat to users of the water supply because of the slow degradative processes in the ground-water



Source: Grisham et al., 1986

Figure 1: Routes of Transport of Hazardous Substances

environment. In addition, ground water is rarely treated prior to human consumption. Hazardous wastes released into surface water either by surface runoff or outcropping of ground water are generally mixed, diluted, and degraded much more readily than in ground water. However, the flowing nature of surface water may lead to long range distribution of chemicals in drinking water supplies or recreational areas so that more people are potentially exposed.

Toxic vapors and particulates released to the atmosphere are subject to atmospheric dispersion. Depending on the wind conditions, temperature and rainfall, hazardous materials can travel potentially great distances before being deposited on land or degraded. Human exposure may occur via direct inhalation of the vapors or particulates. In addition, toxic materials deposited on land may also accumulate in the soil so that uptake by plants can occur. Animals may be exposed to hazardous substances by direct inhalation of the vapor or particulates or by consuming contaminated plants, soil, water, or other animals. Human exposure could occur via the ingestion of either crops or animals which contain bioaccumulated hazardous chemicals. An additional complicating factor for humans is that crops and animals raised for sale are often transported great distances before being consumed by humans, thereby significantly increasing

the distances over which human exposure to contaminants released from disposal sites can occur.

Human exposure to chemicals released from disposal sites may occur by a variety of mechanisms and through various pathways, depending on the location of the disposal site and the management practices employed. The Ontario Ministry of the Environment (MOE) has conducted a waste site inventory, which indicates that there are 69 active and 88 closed landfills that have received liquid industrial or hazardous waste. MOE has stated that these 157 sites pose the highest hazard to human health based on the following criteria (MOE, 1986):

- existing dwelling/structure onsite,
- private or municipal well within 1000 m of the site,
- perennial stream onsite or within 1000 m of the site and there is known domestic consumptive or in-stream use of the stream water within 5 km downstream,
- likely future development of land in urban area or in developed rural area,
- significant aquifer on surface or within 10 m and ground-water movement is downward, or
- past problems at the site.

MOE has stated that the remaining sites indicate a low potential for impact on humans; however, the hazard to the environment may still be high and further investigations may be necessary.

Based on the above criteria, MOE has evaluated the potential for human exposure at these disposal sites in a qualitative manner and concluded that 157 of the sites may pose a threat to human health. For the most part, MOE has not identified individual contaminants at these sites nor have they quantitatively evaluated, through an epidemiological study, the potential for human health effects associated with chemical exposure. An epidemiological investigation has been performed, however, for the Upper Ottawa Street Landfill, a closed landfill located in Hamilton, Ontario. At the time the study was initiated in 1981, the Ontario Ministry of Health stated that this investigation was the first of its kind in Ontario, and thus it was important that it be conducted in a thorough and comprehensive way so that it would be useful as a prototype that could be applied to future problems at other landfills (MOH, 1986).

Epidemiological investigations of populations surrounding waste disposal sites in the United States have been conducted in a number of cases where known environmental contamination has occurred or adverse health effects are suspected. In many cases, definitive evidence linking exposure to adverse health effects has not been found. Investigators conducting such studies, however, face many problems. First, the cost to complete such

studies is very high, making it difficult to obtain the funding necessary to properly conduct a full and complete study. Second, there are difficulties in measuring relevant exposures, in identifying a relationship between the health effect studied and a specific chemical exposure, and in establishing sufficient study sample size in the absence of study confounding and bias. In addition, many studies are conducted in emotionally charged situations and under time constraints. Given the limitations of applying epidemiological study methods, the inability to demonstrate a causal relationship between exposure and health effects to chemicals from disposal sites does not mean that effects do not exist nor that they will not be found in the future.

This chapter presents three case studies in which epidemiological study methods have been successfully used: Upper Ottawa Street Landfill, Hamilton; Love Canal, New York; and Hardeman County, Tennessee. The successful use of epidemiology in these cases indicates that epidemiology is an applicable tool that can work, and which can provide evidence linking waste disposal to adverse human health impacts. These studies support evidence to suggest that there are adverse human health effects associated with exposure to chemicals released to the environment from waste disposal sites.

B. CASE STUDIES

1. Upper Ottawa Street Landfill

The Upper Ottawa Street Landfill is located on the east mountain in Hamilton, Ontario within one kilometre of the face of the escarpment. From the early 1950s until it was closed in 1980, the landfill received many types of domestic and commercial waste, as well as solid and liquid industrial wastes from the Hamilton region, elsewhere in Ontario and from the United States. The landfill now occupies approximately 16 ha. The amounts and types of wastes buried in the landfill will never be known with any degree of certainty, however, it is known from existing records that millions of litres of liquid industrial wastes were deposited at the site during the 1970s.

The waste in the landfill is covered by an intermediate layer of solid waste materials from the local steel industry and a cap consisting largely of clay, which has been seeded with grass. Leachate is collected from some areas of the landfill by a system of pipes and eventually reaches the city sewage treatment plant. Deep erosion gullies have formed on the steep slopes of the landfill, particularly on the north side where leachate flows directly into the creek. Large volumes of methane gas are being produced and vented through the cover and gas vents installed on the south side of the site (MOH, 1986).

The landfill itself sits on fractured bedrock that is in contact with ground water, however, this aquifer is not used as the local drinking water supply. The Red Hill Creek forms the northern boundary of the site. Upper Ottawa Street and the Quinndale neighborhood are on the west side. The neighborhood contains two elementary schools and houses within approximately 500 m of the site. Approximately 615 households are located within 500 m of the landfill, mostly to the west and north of the site. At a distance of 750 m from the landfill there are an additional 960 households (MOH, 1986).

Landfill contamination of the Red Hill Creek has been recognized as a problem for many years. A 1965 report by the Ontario Water Resources Commission concluded that the "gross pollution of the Red Hill Creek appears to have been caused by the unsatisfactory operation of the dump". At that time, the landfill adjoined the creek bank for approximately 365 m, and garbage, burned material and leachate were entering the creek and discoloring the water. A 1968 study by the Hamilton Region Conservation Authority identified the landfill as a serious pollution threat to the Red Hill Creek and recommended that seepage and surface water from the landfill be collected and pumped into a proposed sanitary sewer system. The report also noted that the number of fish and other aquatic species

diminished rapidly in the part of the creek that bordered the landfill. A subsequent study by the MOE in 1978 concluded that landfill pollution of the creek was limited mainly to the organic decomposition of domestic waste, that metals did not appear to be a problem, and that the source of severe bacterial contamination was the landfill (MOH, 1986).

In 1978, the Upper Ottawa Street Landfill Residents Association was formed to combat the continued open burning of waste and the dumping of liquid industrial waste at the Upper Ottawa Street Landfill site. In October of 1980, the Regional Municipality of Hamilton-Wentworth closed the Upper Ottawa Street Landfill because a new alternative facility was developed and because of public concern about possible exposure to toxic chemicals that might be buried there. In this case, the Upper Ottawa Street Residents Association was effective in expressing the concern of its members about possible health hazards associated with living near the landfill. In response to concerns of the public, the Region requested the Ontario Ministry of Health to carry out a health study, and an appointed study committee began its investigation in April, 1981.

In planning an epidemiological investigation to investigate whether or not adverse health effects had been created in

populations exposed to toxic chemicals disposed of in the Upper Ottawa Street Landfill site, the Committee recognized several major obstacles, including: the lack of specific knowledge about the types and amounts of wastes deposited at the landfill and the concentrations of compounds to which persons may have been exposed when the site was operating; ready access for the migration of landfill contaminants in the creek which flows through the site or in the fractured bedrock upon which the landfill is situated; and a "difficult to define" exposed population consisting of a small number of workers, plus residents who, for the most part, did not live in the area until 1976 (MOH, 1986).

Designing health studies of the type needed was hindered because a) there is no standard established methodology for such studies, b) the duration of exposure for most residents is less than 10 years, too short a time for potential chronic effects such as cancer to be detectable, and c) the likelihood of being able to establish a cause-and-effect relationship between illness and the landfill is small because of the problems of isolating landfill exposure from other types of chemical exposure. The Committee conducted separate health surveys of cohorts of workers and residents, which involved questionnaires, the use of control

groups, and medical chart abstraction studies, which included examining the medical charts kept by the family doctors of study participants.

A study of 313 landfill workers and control subjects concluded that three groups of health problems appear to be associated with working at the landfill: chronic bronchitis, daily cough, combined respiratory problems; certain central nervous system symptoms such as headaches, dizziness, lethargy and balance problems; and mood disorders such as anxiety, depression, trouble sleeping, irritability and restlessness. Other identified health problems include difficulty breathing, skin rash, combined skin problems, and muscle weakness in arms and legs. The study also found that the landfill workers were most likely to have developed these health problems between 1970 and 1980, which coincides with the periods of increasing and maximum waste disposal at the site. Furthermore, the landfill workers tended to develop these health problems in the first five years after they began to work at the landfill, which implies that if the landfill is the cause of these problems longer term exposure was not required. An analysis of the overall pattern of mortality for 21 landfill workers who died between 1965 and 1983 indicates that there was no significant difference in the

mortality risk for landfill workers at the time the study was conducted, although this analysis has limited statistical power (MOH, 1986).

The study of landfill residents, which consisted of interviews of 1328 households (5118 people) in the landfill and control populations, had the following results. A positive association was found between landfill residents and certain respiratory, skin, central nervous system (headaches, dizziness, fatigue) and mood (anxiety, depression, irritability) health problems in adult landfill residents. It was also found that adults with longer-term residence in houses located closer to the landfill had a higher incidence of these health problems than did adults living in similar households located farther away from the site, providing strong evidence to support an association between the observed health problems and chemical exposure. There is no evidence to suggest that exposure to the landfill after it was closed and covered in 1980-1981 has led to health problems among area residents (MOH, 1986).

The identified health problems observed in workers and residents are not considered severe in terms of disability at home or in the workplace. However, one important aspect of these findings is that workers and residents were likely to have been

exposed to unknown levels of carcinogenic chemicals at the landfill. No increased incidence of cancer was observed but given the long latency period for development of cancer, final conclusions cannot be reached for at least two decades following the period of exposure. Thus, the Committee recommends setting up a registry of landfill workers and area residents who were identified for the health surveys for the purpose of following the incidence of cancer in these populations.

2. Love Canal

The Love Canal was originally excavated in the 1880s by William Love as part of a scheme to create a sprawling industrial complex between the two branches of the Niagara River. The complex would receive electric power that could be generated by the water's drop from the canal's entrance just above the falls to the Niagara River. The plan for the development eventually collapsed due to advances in the ability to transmit electricity. As early as 1938, a number of private residences were located near the northeast corner of Love Canal. In 1942, Hooker Chemical and Plastics Corporation signed an agreement with the Niagara Power and Development Corporation, owners of the canal, to dump their chemical wastes in the canal.

In 1946, Hooker purchased the Love Canal for use as a chemical waste disposal site. From 1947 to 1952, Hooker dumped over 20 million kilograms of industrial chemical waste into the canal. These wastes included over 6 million kilograms of lindane (benzene hexachloride), a highly toxic and carcinogenic chlorinated hydrocarbon pesticide; over 2 million kilograms of chlorobenzenes, chlorinated derivatives of the highly toxic industrial solvent benzene; and about half a million pounds of trichlorophenol (TCP), which is used in the manufacture of herbicides such as 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), and cosmetic ingredients such as hexachlorophene (HCP), (Epstein et al., 1982).

In April, 1953, Hooker sold the property to the Niagara Falls Board of Education for \$1 in exchange for the Board's signature on a deed with a provision intended to absolve Hooker from all future liability for injury or property damage caused by the dump's contents. In 1955, the 99th Street Elementary School, located adjacent to the eastern edge of the landfill was opened. By 1972, virtually all of the 99 houses whose backyards face the former canal (referred to as the "first ring") were completed, with most of the residential development occurring from the mid-1950s to the early 1970s. As part of the development in this area, storm sewers, roads, and playgrounds were constructed on the landfill site or in the vicinity.

In the 1970s, several problems became apparent to the Love Canal residents: 1) portions of the landfill subsided and drums surfaced in a number of locations; 2) ponded surface water, heavily contaminated with chemicals, was found in the backyards of adjacent houses; 3) unpleasant odors (caused by the volatilization of surfaced chemical wastes) were cited by residents as a factor contributing to both discomfort and illnesses; 4) evidence of subsurface migration of toxic chemicals became apparent in the basements of some neighboring houses with the appearance of a noxious, oily residue accumulating in basement sumps, the corrosion of sump pumps, and the physical evidence of chemical infiltration through cinder-block foundations; and 5) noxious chemical fumes were noticed emanating from several nearby storm sewer manhole covers (USEPA, 1982).

Beginning in 1976, local residents began reporting elevated incidences of a variety of health symptoms. Children were often burned by the liquids oozing from the canal. Neighborhood dogs that spent a lot of time at the canal lost their hair, developed skin diseases, or became sickly and died. Residents were soon reporting more serious effects, including elevated incidences of miscarriages and children with multiple birth defects, severe asthma, and congenital heart defects. Surveys conducted by the New York State Department of Health and Department of

Environmental Conservation between 1976 and 1978 found that in the southern section of the canal, women between the ages of thirty and thirty-four had a miscarriage rate nearly four times higher than normal. The air, water, and soil of the entire canal area were found to be heavily contaminated with a wide range of toxic and carcinogenic chemicals coming from the former Hooker dump. For example, sediment samples from a nearby creek were found to be contaminated with high levels of TCDD, and chloroform was found in the air of one Love Canal home at a level more than one hundred times that considered by the USEPA to cause a significant excess of cancer risk (Epstein et al., 1983).

In 1978, the Love Canal Home Owners' Association conducted its own preliminary health survey of over 100 residents by telephone. Although the study was not scientifically conducted (it lacked a proper control group and likely evidenced response bias), it showed an increased reporting of certain problems in the most exposed households. These problems included asthma, urinary tract problems (incontinence, infections, strictures, and renal failure), central nervous system disorders (dizziness, seizures, blurred vision, hyperactivity in children, suicides, suicide attempts, and nervous breakdowns), and adverse reproductive outcomes (miscarriages, stillbirths, and birth defects) (Paigen, 1982).

After a review of all available data, the Commissioner of the New York State Department of Health declared a health emergency at Love Canal on August 2, 1978, which resulted in the closing of the 99th Street school and a recommendation for the temporary evacuation of pregnant women and all children under the age of two who resided in the first two rings of houses surrounding the canal. On August 7, 1978, the Governor of New York State announced that the State would purchase all houses immediately adjacent to Love Canal (USEPA, 1982). This was subsequently expanded to include all 238 houses in the first and second rings around the canal. In the latter part of 1978 and through 1979, remedial actions such as construction of a barrier drain system, leachate collection and treatment, and construction of a clay cap over the landfill were completed.

In January 1980, at the request of the United States Department of Justice who had filed a civil lawsuit against Hooker, the United States Environmental Protection Agency (USEPA) began a cytogenetic assessment of 36 Love Canal residents to determine if excess chromosome damage was present among Love Canal residents, and if the prevalence and severity of cytogenetic abnormalities warranted a full-scale investigation. The results of the study suggested that additional testing was warranted, since eleven of the 36 Love Canal residents surveyed

had major chromosomal abnormalities (Epstein et al, 1982). The results of this study were quite controversial since proper controls were not analyzed concurrently.

A study conducted in May 1980 by Paigen et al. (1985) examined the health of Love Canal children, since children were believed to be more susceptible to the effects of many toxins and are not directly exposed to substances such as cigarette smoke, alcohol and occupational chemicals. The study population consisted of 523 Love Canal children and 440 control children in two groups -- homeowners and renters. The findings of the study indicated an increased prevalence of seven health problems among Love Canal children compared to the controls after adjusting for age, race, sex, household size, income, and education: seizures, learning problems, hyperactivity, eye irritation, skin rashes, abdominal pain and incontinence. Each of these problems shows evidence of a dose-response relationship using either distance of homes from the canal or proximity of homes to possible paths of chemical migration (referred to as wet areas since these paths were located along old stream beds), thereby strengthening the association of these problems with residences in the Love Canal neighborhood.

An additional study conducted by Paigen et al. in June and July, 1980, examined the height and weight of the same children in order to determine whether living near a hazardous waste site had an adverse impact on growth (Paigen, et al., 1986). In order to standardize for sex, race, and age, percentiles were assigned to the anthropometric measurements. Using statistical analyses it was found that children born and spending at least 75% of their lives in the Love Canal area had significantly shorter stature for age than control children (mean stature for age percentile of 46 ± 2.2 compared to 53.3 ± 1.4 for controls). The difference in stature associated with birth and residence in the Love Canal neighborhood could not be accounted for by differences in midpoint height, socioeconomic class, nutrition, birth weight, or chronic illness. It was also found that there was no difference in weight for stature percentiles (mean weight for age percentile was 50 ± 2.2 for exposed children compared to 53.1 ± 1.4 for controls) thus indicating that stature is more affected than weight.

Two studies, one by Vianna (1980) and one by New York State Department of Health (1981) as cited in Grisham, 1986, were initiated to investigate the incidences of spontaneous miscarriages and of low birth weight. The authors found no significant excess of congenital defects in the study area with

the possible exception of "wet area" homes when compared to "dry area" homes. In addition, in the study area as a whole no significant excess of spontaneous abortions were observed, although a significant excess was observed in the "wet area" homes when compared with the "dry area" homes. Also, a higher proportion of low birth weights was recorded in the "wet area" than in the "dry area". Doubts exist as to the use of these endpoints as reliable measures of adverse health effects and, with the exception of birth weights, which were checked by referring to birth certificates, the study was limited because it relied on recall.

A number of additional health studies have been conducted at the Love Canal. One study by Janerich et al. (1981) as cited in Grisham (1986) concluded from an examination of data in the New York State Cancer Registry that there was no evidence of higher cancer rates in the area close to Love Canal compared to upstate New York rates. In another study by Heath et al. (1983) as cited in Grisham (1986), no significant differences in the frequency of chromosomal aberrations or sister chromatid exchange were found between residents living close to the Love Canal and in other parts of Niagara Falls. Both of these results are not, however, unexpected. As noted in the previous chapters, significant lag times exist between exposure and the manifestation of chronic

disease such as cancer. Given the relatively short time that has passed since exposure of the Love Canal population, the failure to find an increased incidence of cancer may reflect the fact that the necessary lag time has not elapsed. Only a study properly conducted after an appropriate lag time can determine whether or not the incidence of cancer in the Love Canal population is increased. Similarly, the timing of the chromosomal abnormality study is also questionable. At the time of the study, the exposure of Love Canal residents had been abated for some period of time. The types of adverse effects looked for would only be expected to be present, if they were to be present at all, at times immediately following exposure and not after a significant period of non exposure.

In the Love Canal case, there was visible proof of the danger of acute exposure (chemical burns received by children playing in the school yard). In addition, several studies suggest the existence of a direct cause-and-effect relationship between harmful effects and chronic, off-site, low-level exposure. Not all the studies performed support this conclusion and a number of criticisms have been raised about the studies that do and do not support a cause-and-effect relationship between exposure and the incidence of adverse health effects. Taken as a whole, however, the Love Canal experience supports the

premise that improper hazardous waste disposal can lead to the release of chemicals in the environment and the induction of adverse human health effects.

2. Hardeman County Waste Disposal Site

Velsicol Chemical Corporation began hazardous waste disposal operations on an 8 ha portion of a 98 ha farm off Toone-Teague Road in rural Hardeman County, Tennessee in 1964. The waste included the residues from the manufacture of heptachlor, dieldrin, endrin, and heptachlor epoxide. In addition to these pesticides, the wastes contained chlordene, isodrin, tetrachloroethylene, hexachlorocyclopentadiene, hexachlorobicycloheptadiene, carbon tetrachloride, and chloroform (Sprinkle, 1978).

Hydrogeological studies conducted by the United States Geological Survey (USGS) in 1967 concluded that although contamination of surface waters and of shallow ground water had already taken place, ground water was flowing in a direction away from the local private drinking water wells (Rima et al., 1967). Relying on the USGS report that indicated there was no danger to the local drinking water wells, Velsicol expanded the landfill from 8 to 18 ha (Epstein et al., 1982). In 1972, the state ordered a halt to the dumping; however, disposal operations

continued into 1975. By this time, over 300 000 200-litre drums of solid and liquid pesticide production wastes had been buried in shallow trenches (Epstein et al., 1982).

By 1977, local residents were complaining of taste and odor problems in their well water and reporting an unusually high incidence of health symptoms, which included skin and eye irritation; weaknesses in the upper and lower extremities; upper respiratory tract infection; shortness of breath; and severe gastrointestinal symptoms, including nausea, diarrhea, and abdominal cramping (Clark, et al., 1982). Unfortunately, the 1967 hydrogeological studies had incorrectly predicted the direction of ground water flow and had failed to consider changes to the ground water system due to expansion of the dumping area, increased use of water, and encroachment on the site by housing (Epstein et al., 1982). Hence, by 1978 pesticide wastes had contaminated drinking water wells with extraordinarily high concentrations of a number of known carcinogens (e.g., carbon tetrachloride, chloroform, and tetrachloroethylene) and other substances that may represent a chronic health threat. Most residents stopped using the well water for drinking water in 1978, but use for showering, cooking, etc. continued into 1979.

Extensive ground water sampling has been conducted by the USEPA and the State of Tennessee since 1978. By late 1978, over 12 chlorinated organic compounds in the wells serving individuals around the site had been detected. Concentrations of carbon tetrachloride, the contaminant found in highest concentrations ranged up to 18 700 ug/L in one private well approximately 550 m to the north of the landfill (Clark et al., 1982). As is usually found with ground water contaminated from hazardous waste landfills, the concentrations fluctuated over an approximate ten-fold range during the year. Subsequent water quality data indicated increasing concentrations of most contaminants with time. For example, in one domestic well approximately 460 m north of the landfill, the concentrations of carbon tetrachloride ranged from 9820 ug/L to 20 000 ug/L from November 1978 through November 1979; and from 18 000 ug/L to 164 000 ug/L from May 1981 through June 1982 (Orr et al., 1982).

A health survey was conducted by Clark et al. in 1978 to determine whether exposure to the contaminated water was associated with liver dysfunction. The 151 individuals studied were divided into three groups based on their level of exposure: 61 individuals who used water with carbon tetrachloride concentrations greater than 150 ug/L; 33 individuals exposed to carbon tetrachloride at concentrations less than 45 ug/L; and an

unexposed control population of 57 individuals. The initial hepatic profile revealed elevated concentrations of the serum enzymes, alkaline phosphatase and serum glutamic oxaloacetic transaminase (SGOT) in residents who used the water with carbon tetrachloride at both concentrations (Clark et al., 1982). During physical examination, seven individuals (six from the high exposure group and one from the intermediate exposure group) had borderline liver enlargement.

In an independent survey of residents in 1982 by Rhamy, physical examinations of 102 residents showed 28% with diastolic hypertension; 8% with increased liver size; 2% with borderline liver enlargement; 7% with optic atrophy; 4% with borderline optic atrophy; 10% with peripheral neurological changes; 8% with borderline peripheral neurological changes; and 32% with significant eye problems. Although a control group was not included in this study, Rhamy concluded that the extraordinarily high incidence of abnormal physical symptoms and clinically observed abnormalities were most likely related to the chemical exposures from the contaminated ground water (Harris et al., 1983). Rhamy also considered the high incidence of eye problems, particularly optic atrophy, to be noteworthy, since optic atrophy has been associated with occupational exposures to carbon tetrachloride (Smith, 1950). It should be noted that it is very

difficult to adequately assess findings of a study in the absence of a control group and in the absence of demographic information such as the age distribution and method of selection of the study population.

An analysis by Harris et al. (1983), compared the findings of these health surveys with the predicted adverse health impacts suggested from human exposure estimates and toxicological studies on laboratory animals. In this study, exposure estimates for four chlorinated organics (including carbon tetrachloride) were calculated for a male infant and an adult male who lived in a house served by a well located approximately 550 m north of the northern boundary of the landfill. It was found that the estimated average daily exposure to carbon tetrachloride for the infant exceeded the minimum effective dose that produced liver damage in guinea pigs, and that the estimated adult exposure was approximately 20% of the minimum effective dose. The study concluded that Toone-Teague residents were at high risk of liver damage, considering the presence of a number of other liver toxins such as chloroform and tetrachloroethylene that were present in the water but not included in the risk estimate.

On August 1, 1986, the United States District Court for the Western District of Tennessee entered a judgment against Velsicol

Chemical Company in a class action for polluting plaintiffs' drinking water. Five plaintiffs received over \$5 million total compensatory damages and \$7.5 million was levied for punitive damages. Approximately 120 other plaintiffs await the trial of their damage claims. The compensatory damages consisted of varying amounts of money for increased risk of cancer, actual physical injury, impairment of plaintiffs' immune system, psychiatric damage, physical pain and emotional suffering and impairment of the quality of life.

V. CONCLUSION

It is clear that a large number of chemicals present in special wastes are known or suspected to produce adverse health effects in exposed humans. Improper disposal of such wastes often leads to the release of the chemicals to the environment so that the potential for human exposure exists. Epidemiological investigations provide the basis for determining whether or not exposure to the chemicals released into the environment has resulted in adverse human health effects in the surrounding community.

The Upper Ottawa Street, Love Canal, and Hardeman County case studies indicate that a link exists between the improper disposal of special waste and specific human health effects in populations surrounding disposal sites. However, epidemiological investigations of populations surrounding disposal sites are often unable to establish that a direct relationship exists between inadequate disposal practices and adverse human health consequences. The difficulty of establishing such a relationship may be associated with limitations in available data or problems

in establishing accurate exposure scenarios. These problems are compounded in studies that attempt to establish such relationships in the general population as opposed to those living in proximity to the disposal site. Attempts to link health effects to human exposure of hazardous chemicals on a larger scale are difficult if not impossible to establish and consequently, epidemiologists most often focus their investigations on populations surrounding the disposal site. The failure to obtain positive results at the time of the study, however, may not be convincing evidence that the chemical exposure has not caused adverse health effects in the exposed population. Toxicological data can provide a warning of the potential hazard posed by chemicals released into the environment before exposed populations develop illnesses as a result of inadequate disposal practices.

The concentration of hazardous wastes in disposal sites has the potential to seriously harm the environment and health of populations living near the disposal sites if the wastes are not properly managed. Industrial societies will continue to generate hazardous wastes and there will continue to be advances in production processes and practices that reduce hazardous waste volumes and reuse waste byproducts. Hazardous waste management facilities that are properly located, use appropriate

technologies, and are optimally managed provide the key to minimizing the release of hazardous contaminants to the environment and thus minimizing the potential for adverse human health impacts.

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